OBSERVATIONS ON FIFTY-FOUR CASES OF LOCOMOTOR ATAXIA, WITH SPECIAL NOTES ON ETIOLOGY.[1]

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Tabulated symptoms of fifty-four cases:
Lost knee-jerk, 88%.
Romberg's symptom, 80%.
Ataxic gait, 77%.
Lightning pains, 73%.
Paresthesia, 70%.
Argyll-Robertson pupil, 67%.
Incoördination, 65%.
Vesical disturbance, 60%.
Paralysis of ocular muscles, 27%.
Sexual weakness, 25%.
Diplopia, 21%.
Girdle sensation, 19%.
Skin reflexes disturbed, 14%.
Ptosis, 11%.
Muscular sense lost, 10%.
Arthropathies, 8%.
Perforative ulcers, 6%.
Crises, 4%.
Muscular atrophy, 4%.
Optic atrophy, 2%.
Nystagmus, 2%.

Of the symptoms tabulated, the six most frequent were, in order mentioned: Lost knee-jerk, static ataxia, locomotor ataxia, lightning pains, paresthesia, and the rigid pupil to light. Closely following these were: Incoördination, bladder difficulties, and ocular palsies. The lost knee-jerk, the Argyll-Robertson pupil, ataxia, and the lightning pains furnish the classical symptom-complex of tabes. The presence of any three of them makes a diagnosis clear; or, the finding of any two with one or two signs and symptoms of minor significance, such as bladder weakness, or diplopia, justifies a diagnosis of tabes.

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[1] Read at Calhoun County Med. Asso., Aug. 13, 1901.

The order of onset of symptoms varied greatly. Difficulty in walking in the dark, or through a crowded thoroughfare usually first awakened the patient's interest. Often, pains designated "neuralgic" and "rheumatic" preceded the ataxia. Paresthesia was common, being present in 70% of cases, and was often the first symptom. In five or six cases, difficulty in starting the urine, or sphincter weakness, was the first symptom. In four cases, double vision was the initial symptom. In one case gastric crises ushered in the disease.

The conditions which in our experience most often need differential exclusion are certain forms of peripheral neuritis, neurasthenia, spinal syphilis, and early general paresis. The importance of safely excluding the latter graver disease leads me to cite a case, and to discuss briefly the supposed relation of tabes and paresis.

Two years ago a patient in the third decade of life applied for treatment. A diagnosis of tabes was made. He had a lost patellar reflex; his pupil responded to accommodation, but not to light; he had ataxia and gastric crises. Other signs and symptoms were absent. He took treatment several weeks, and was advised to return in a year, which he did. Considerable improvement was noted in walking, and the gastric crises had ceased. One week before his third return, mental symptoms of an exalted nature, with delusions of grandeur suddenly developed. This elation and expansion have largely subsided, and the patient is mentally reduced. The diagnosis is now, undoubtedly, general paresis.

The interesting question is, Was it general paresis two years ago? The German and French schools say that in a certain measure every case of tabes is one of incompletely developed paresis. The majority of neurologists, however, see in paresis an independent disease which is developed in tabetic individuals. According to Fürstner, in 62% of paretics there is found a disease of the lateral and posterior columns of the cord. Accordingly the very earliest signs which may precede the beginning of the abnormal psychical manifestations are those belonging to the symptom-complex of locomotor ataxia—Argyll-Robertson pupil, lost knee-jerk, lancinating pains, incoördination, and bladder weakness. So universally are physical signs present in paretics, that today one would scarcely care to diagnosticate paresis without the presence of some of the cord-symptoms usually found in tabes.

About a dozen cases—not in this series—were differentiated from tabes before the onset of mental symptoms only by the characteristic clumsiness of speech and tremor of the tongue and muscles of the lower face. In other respects they resembled tabes closely. Two of these patients have within the past year developed the typical mental symptoms of paresis. It can be safely advanced that tremor of the tongue and lips, and changes in articulation, with perhaps mild changes in the disposition of the patient, change the complexion of the diagnosis to the graver malady, general paresis. An early diagnosis of locomotor ataxia is of great importance. The physician's skill in detecting the malady early offers the patient about his only hope of relief.

The prognosis, judging from results obtained with these fifty-four cases, is not altogether bad. Improvement in the general health is almost always observed, and usually those who come in the first, or even the second stage, have the disease processes stopped or delayed for an indefinite period.

Etiology of tabes—The disease is much commoner in men than in women. Of the fifty-four cases presented, two were women. Hereditary influences are rare. Exposure to wet and cold; fatigue; excesses, particularly sexual, are considered predisposing causes; excessive smoking; and the continuous moderate, or the occasional excessive use of alcohol, invite degenerative processes. Fifty-four per cent. of this series gave histories of tobacco or alcoholic dissipation.

The toxins of infective fevers are undoubtedly capable of reducing the resistant powers of the individual, and occasionally play the rôle of exciting causes. Eleven per cent. of these cases followed influenza, and in four per cent. the symptoms of tabes appeared immediately after typhoid fever; four per cent. received a trauma to the spine a few months before the onset of symptoms. Almost all cases developed between the ages of thirty and sixty. In five, the symptoms first appeared in the third decade—a small percentage in comparison to other series—sixteen in the fourth decade; nineteen in the fifth; seven in the sixth; and two reported first symptoms as late as the seventh decade. I have a patient now in his eightieth year, the diagnosis of whose malady I am yet in doubt, but whose findings point strongly to tabes.

Less importance is now being given to syphilis as a causative agent than formerly; or, at least, greater attention is given to the possible rôle of other poisons. Three distinct positions are taken regarding the existance of a causal relation between syphilis and tabes. The position of extreme affirmation is occupied by Ferrier, and others whose opinions may be briefly expressed, "No syphilis; no tabes." At the other extreme stand Leyden and others who either deny the relation altogether, or hold that it is still unproved. Of Leyden's 108 cases,[2] only twenty per cent. were syphilitic—a percentage not greatly above that found in non-tabetic patients.

Grimm[3] draws attention to the fact that syphilis is rife and virulent among the Japanese and negroes, and tabes rare. Lewin is cited as finding no case of tabes in 800 female syphilitics. Gluch found an absence of tabes in 3,000 syphilitic Bosnians.

Drennen[4] suggests a possible factor in producing locomotor ataxia in the long continued and uninterrupted administration of large or even measurably large doses of iodide of potassium which is so commonly given at the present day; and other neurologists are seriously considering this possibility.

A middle position is assumed by a great majority of observers who maintain that 45 to 80 per cent. of cases of locomotor ataxia are due to syphilis. Of our cases, including the positive specific, and four doubtful cases, 42 per cent. were syphilitic. But while syphilis is an antecedent in a large percentage of cases, syphilitic cases resemble the non-syphilitic as regards the histological characters of the lesions, and specific treatment has no effect upon cases with syphilitic history. Hence the broadest conclusion which we can deduce from the fact is that syphilis alters the physiological condition in such a way as to favor the attack and operation of the actual cause of tabes. In this its influence is like that of measles, or scarlet fever, in leading to the development of tuberculosis; and similar, also, to the action of the influenza bacillus in relation to other pathogenic organisms.

I quote from a recent writer: "When to these facts of clinical experience, we add the evidence of experimental bacteriology, from which we learn, for example, that the presence of staphylococci and streptococci leads to a much more luxuriant growth of the influenza bacillus; and, further, that the latter will grow more rapidly in a medium through which these other organisms have passed, we are strangely attracted to the existence of an analogy in the relation of the syphilitic virus to the toxic agent responsible for locomotor ataxia."[5]

Thus from the many factors attributed as the cause, and the lack of uniformity in the estimation of value of the various supposed etiological agents, it is evident that the pathogenesis is not fully understood, and that in quite a large percentage of cases causes are at work preparing soil for degenerative processes in the spinal cord other than syphilis. Very pertinent and instructive observations and experiments have been made recently, and are now being conducted, with the end in view of determining what these influences are.

In reviewing the fifty-four cases of locomotor ataxia of this paper, surprise was elicited at the large percentage of patients giving a definite history of gastro-intestinal disturbances, such as army diarrhea, gastric and intestinal catarrh, chronic gastritis, chronic constipation, gastroptosis and enteroptosis, and chronic liver troubles, antedating the onset of any of the symptoms of tabes from a few months to several years. Forty-six patients, or 85 per cent. of cases, gave such histories.

Chalmers Watson[6] obtains conclusions based upon experimental work and clinical study, which are pertinent to the large percentage of gastro-intestinal disorders preceding tabes in this series. Fowls fed on a diet limited to red meat and water, developed symptoms of an acute nature, ataxia, paraplegia, gradually developing unconsciousness and death. Others exhibited no nervous symptoms. In a third class severe attacks occurred of a nature similar to those of the first group, without, however, involvement of the higher neurones. At the time of the attacks no change was made in the dietetic regime, the only difference being that the animal ate sparingly. A slow recovery occurred on each of these occasions which was probably due to the acquisition of some degree of immunity to the action of the toxic substance or substances responsible for the symptoms.

In two clinical cases of tabes, Dr. Watson has observed initial symptoms, directly referable to the digestive tract, and regards the presence of these gastro-intestinal lesions of primary importance in the production of lowered power of resistance to toxic influences leading to a later development of the spinal cord lesions.

Treatment—Our opinion is that there is no therapeutic agent of as much value in successfully combating the degenerative processes, and in raising the vitality of the diseased parts and of the entire body, as hydrotherapy. Winternitz, after a long experience, gives his results of treatment of 1,000 cases treated by hydrotherapy as being vastly more satisfactory than by any other mode of treatment. The general indication as well as indications for the treatment of special symptoms such as, for example, the cutting pains, are more uniformly and satisfactorily met by scientific applications of hydrotherapy than by medicine.

Extremes in temperature are to be avoided, as well as excessive corporeal exercises. Rest is one of the first and important indications. Electricity and massage give good results.

The suspension treatment as formerly given was, in our judgment, too severe; and while good results were observed in cases, we no longer use the suspension apparatus, but obtain equally satisfactory results by stretching the cord through lengthening the vertebral column, thereby inducing a mild inflammatory reaction—the end sought by the suspension treatment. (In this method, the patient is seated upon a table and the legs are held in an extended and adducted position, and the upper part of the body is then forcibly flexed forward.)

One of the later methods of treatment of tabes has produced, in our experience, since its adoption, very satisfactory results with patients treated. This consists in the methodical exercises by which the patient is gradually re-taught coördinated movements. The idea was first advanced and recommended by Fraenkel, and has received much commendation by German neurologists. Tabetics have lost in part the power to properly execute and coördinate fine movements. These exercises require the patient to pass from simple to complicated movements. He is made to relearn the control exercised by his eyes and the rest of his sensory apparatus, the proper motor impulses which induce coördinated movements. That it is of much value is without question. A patient left the Sanitarium last week, who, upon his arrival, could neither walk nor stand even with the help of crutches; and who gradually relearned the art of walking so that he could walk nicely with a cane. Another patient, apparently in the third, or paralytic stage, bed-ridden upon arrival, left, walking with a cane and crutch. He continued the exercises and returned a few months ago without the crutch. At the present time he is disabled by a Charcot's joint.

In a word we may say that in the last 24 or 25 patients of this series, who have been given the Fraenkel exercises, very satisfactory results have been obtained. It is not an unusual experience for crutches and canes to be laid aside. I do not give all the credit to this system, by any means; but will say that the treatment of tabes is by no means complete without its careful and persistent use. Each patient is given a type-written schedule of exercises which he follows carefully every day, once or twice, under the direction of an assistant.

We refer again to the chronic gastro-intestinal disturbances of severe character antedating the onset of tabes. Eighty-five per cent. gave a history of such marked disturbances against 42 per cent. who gave a history of syphilis. It is difficult to resist the conclusion that the above chronic disorders of the intestinal tract produced disturbances of nutrition of primary and direct importance; and that the production of toxins and poisons within the alimentary tract, and their absorption and presence in the general circulation would interfere with the nutrition of the cord, and that defective nutrition accounts simply and adequately for degeneration of the nerve fibers.

One of the first indications, therefore, for treatment in the light of the experimental work upon the effect of a toxic diet upon fowls above quoted and supported by the experience of Dr. Watson, and also by our own cases, would be the treatment of the gastro-intestinal disturbances, and the establishment of a healthy condition of the digestive tract, by giving the patient a proper and nourishing diet. Along this line we with others have observed that gastric and rectal crises, and the lancinating pains of tabes are often concomitant with, and preceded by, a dirty tongue and constipated bowel. Elimination and kidney function are to be increased.

I again quote from the *British Medical Journal:* "I submit that the proper line of investigation is directed to the discovery of the nature and source of the toxic substance at work. At the same time we must study the clinical facts of the disease in the light of the results obtained by experimental bacteriologists and others investigating the all-important subject of immunity. In my opinion the alimentary tract furnishes the chief area of investigation, and in all probability it will be proved to be the original source of the toxemia. In this connection, it is interesting to refer to the experimental work of Adami and others on the different forms of chronic infection, due primarily to the presence in the tissues of bacterial poisons derived from the intestine. The diseases especially investigated by Adami were cirrhosis of the liver, hemachromatosis, and pernicious anemia; and this author as quoted by Putnam and Taylor, suggested that this subinfection would be found to play a definite part in the development of many chronic fibroid conditions."

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[2] Lyons Méd., '95.
[3] Inter. klin. Rund., Aug. 29, '94.
[4] Alienist and Neurologist, Oct., '96.
[5] British Med. Jour., June 1, 1901.
[6] British Medical Journal, June 1, 1901.